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Differences in Cardiac Parameters among Elite Rowers and Subelite Rowers

AARON L. BAGGISH¹, KIBAR YARED¹, RORY B. WEINER¹, FRANCIS WANG², ROBERT DEMES³,
MICHAEL H. PICARD¹, FREDRICK HAGERMAN⁴, and MALISSA J. WOOD¹

¹Division of Cardiology, Massachusetts General Hospital, Boston, MA; ²Harvard University Health Services, Cambridge, MA;
³United States Army Research Institute for Environmental Medicine, Natick, MA; and ⁴Department of Biomedical Sciences,
Ohio University, Athens, OH

ABSTRACT

BAGGISH, A. L., K. YARED, R. B. WEINER, F. WANG, R. DEMES, M. H. PICARD, F. HAGERMAN, and M. J. WOOD. Differences in Cardiac Parameters among Elite Rowers and Subelite Rowers. *Med. Sci. Sports Exerc.*, Vol. 42, No. 6, pp. 1215–1220, 2010. There is significant individual variability in the cardiac adaptation that occurs in response to exercise training. Factors associated with this variability remain incompletely understood. To date, the relationship between the competition level at which athletes participate and their underlying cardiac parameters has not been explored. **Purpose:** The purpose of this study was to determine whether parameters of cardiac structure and function differ significantly among elite competitive rowers (ER), subelite competitive rowers (SR), and sedentary controls (C). **Methods:** Cardiac parameters were assessed in ER ($n = 20$), SR ($n = 20$), and C ($n = 20$) using two-dimensional, tissue Doppler, and speckled-tracking echocardiography. **Results:** Physiologic cardiac remodeling was present in both ER and SR as evidenced by the significant differences in the majority of structural and functional parameters in both rower groups when compared with C. When compared with SR, ER were found to have greater left ventricular (LV) end-diastolic volume (76 ± 6 vs 71 ± 8 mL·m⁻², $P = 0.02$), LV mass (150 ± 11 vs 134 ± 16 g·m⁻², $P = 0.002$), and right ventricular (RV) end-diastolic chamber dimensions (15.6 ± 0.9 vs 13.9 ± 1.5 cm²·m⁻², $P < 0.001$). Further, ER demonstrated significantly more enhancement of RV systolic function (peak strain = $36\% \pm 7\%$ vs $31\% \pm 6\%$, $P = 0.008$) and late diastolic relaxation in both the LV ($A' = 4.2 \pm 1.3$ vs 3.2 ± 0.9 cm·s⁻¹, $P = 0.01$) and the RV ($A' = 6.6 \pm 1.4$ vs 4.3 ± 1.3 cm·s⁻¹, $P < 0.001$). **Conclusions:** Although cardiac remodeling occurs in both ER and SR, specific aspects of cardiac structure and function differ between rowers who compete at the elite and the subelite levels of sport. **Key Words:** EXERCISE-INDUCED CARDIAC REMODELING, ATHLETE'S HEART, ELITE ATHLETE, MYOCARDIAL FUNCTION, DIASTOLIC FUNCTION

Exercise training leads to adaptive changes in cardiac structure and function. Left ventricular (LV) hypertrophy and dilation (6,26), right ventricular (RV) dilation (9,35), and left atrial enlargement (11,29) have been demonstrated in trained athletes. Recently, corollary changes in cardiac function have also been reported (4,7,40). Close examination of the extensive prior cross-sectional data (27,30,31,36) and recent longitudinal studies (2,8,23) demonstrate significant interindividual variability in the cardiac structural and functional response to exercise. Although contributory factors including age (32), gender (28), sport type (21,39), ethnicity (5), and gene profile (10,20) have

been identified, the variability in cardiac morphology and function among athletes remains incompletely understood.

There are convincing animal data which demonstrate that myocardial cell adaptations to exercise are dose dependent (12–14). Although recent human data suggest a dose–response relationship between exercise and clinical outcomes (16,37), the relationship between exercise exposure and cardiac remodeling in humans remains unknown. The volume and the intensity of exercise training typically parallel increases in the level of competition, and it is plausible that the magnitude of exercise-induced cardiac remodeling may follow suit. Although cardiac remodeling has been demonstrated in elite, subelite, and recreational athletes, we are unaware of any prior studies that provide a direct comparison of single-sport athletes at different competitive levels. We hypothesized that elite competitive rowers (ER) would have significant cardiac differences, including ventricular chamber dimensions, mass, and diastolic function, when compared with rowers participating at a lower competition level. To test this hypothesis, we conducted a comparative analysis of ER, subelite competitive rowers (SR), and sedentary controls. The primary objective of this study was to determine which structural and

Address for correspondence: Aaron L. Baggish, M.D., Massachusetts General Hospital, Yawkey Building, Suite 5B, 55 Fruit St., Boston, MA 02114; E-mail: abaggish@partners.org.

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functional cardiac parameters differ among rowers at different levels of competition.

METHODS

Study participants. The ER group was comprised of members of the United States Olympic rowing program (U.S. Rowing, Princeton, NJ). ER were studied at a team training facility during preparation for the 2008 Beijing Olympic Games after 3 months of organized team training. The SR group was composed of university student athletes (Harvard University, Cambridge, MA) who were members of the varsity rowing program and were studied after an autumn semester (approximately 3 months) of organized team training. SR were eligible if they were single-sport athletes who participated only in team-based rowing training. Sedentary controls were university students with no history of cardiovascular disease who engaged in minimal formal exercise training ($<1 \text{ h} \cdot \text{wk}^{-1}$). Written informed consent was obtained from all participants before involvement. The Partner's Healthcare Human Research Committee approved the protocol before study initiation.

Height, weight, resting vital signs, medication use, and family history of hypertension as previously defined (3) were recorded at the time of enrollment. Data characterizing short- and long-term exercise exposure were collected from each participant at the time of enrollment and were confirmed with their respective coaching staff. Recent, short-term exercise training exposure was defined as rowing training volume ($\text{h} \cdot \text{wk}^{-1}$) during the 8 wk before enrollment. Long-term exercise training exposure was defined as the number of years of participation in organized competitive rowing at the high school level or above.

Echocardiography. Two-dimensional transthoracic echocardiography was performed using a commercially available system (Vivid-I; GE Healthcare, Milwaukee, WI) with a 1.9- to 3.8-MHz phased-array transducer. Images were obtained after 10 min of quiet rest and were performed $\geq 12 \text{ h}$ after the previous training session. Two-dimensional, pulsed-Doppler, and color tissue Doppler imaging from standard parasternal and apical transducer positions were performed. A two-dimensional frame rate of $25\text{--}75 \text{ s}^{-1}$ and a tissue Doppler frame rate of $>120 \text{ s}^{-1}$ were maintained for all images. All data were stored digitally, and measurements of structure, tissue velocity, and strain were performed by study cardiologists blinded to subject group (EchoPac, Version 6.5; GE Healthcare).

Two-dimensional measurements were made in accordance with current clinical standards (15). All structural measurements are reported as unadjusted and body surface area (BSA)-adjusted values. LV ejection fraction, end-diastolic volume, and end-systolic volume were calculated using the modified Simpson's biplane technique. RV fractional area change, a validated index of RV function, was calculated by outlining the endocardial borders of the RV in diastole and systole in the apical four-chamber view and calculating the

difference between the two areas expressed as a percentage of end-diastolic RV area (1). LV mass was calculated using the area-length method (15). Peak longitudinal LV and RV tissue velocities during systole (S'), early diastole (E'), and late diastole (A') were measured offline from two-dimensional color-coded tissue Doppler images and were reported as the average of three consecutive cardiac cycles. Tissue Doppler velocities were measured from an apical four-chamber view using a $6 \times 2\text{-mm}$ region of interest placed in the basal lateral LV wall. The ratio of the transmitral E-wave velocity to E' was used as an approximation of left atrial pressure (22). LV strain was measured by speckled-tracking analysis (EchoPac, Version 6.5; GE Healthcare) in the apical four-chamber view and is reported as the average of the six LV segments (basal, midventricular, and apical segments of the interventricular septum and the lateral LV wall). RV strain was measured by tissue Doppler analysis from the apical four-chamber view by placing a $6 \times 2\text{-mm}$ region of interest in the mid-RV free wall at a location half way between the apex and the tricuspid annulus at end diastole. The location of this region of interest was manually adjusted in each systolic frame to ensure optimal tracking of the mid-RV free wall throughout RV systole. The reported value is the average of three consecutive cardiac cycles.

Statistical analysis. Measurements are presented as mean \pm SD. Comparison of data across groups was performed using one-way ANOVA. Post-ANOVA, paired t -testing with Bonferroni correction was used for between-group comparisons. Data analysis was performed using the Statistical Package for the Social Sciences for Windows (Version 16.0; SPSS Inc., Chicago, IL), and significance was set at a $P < 0.05$ for all calculations.

RESULTS

Demographic data are presented in Table 1. All participants were Caucasian males. Both groups of rowers (ER and SR) were heavier than the controls, and ER were taller than both SR and controls. Resting heart rate was significantly lower in both groups of rowers than that in controls. Diastolic blood pressures were significantly and similarly lower in both ER and SR when compared with controls, whereas systolic blood pressure and the prevalence of familial hypertension were similar in all groups. The prevalence of prescription medication use, limited to topical antibiotics, antidepressants, and bronchodilator inhalers, was similar in all groups. As expected, ER had accumulated more years of rowing experience than SR and had performed more training during the 8-wk period before study measurements than SR.

Rowers versus Controls

Cardiac structure. Rowers in both study groups (ER and SR) had significantly greater cardiac structural dimensions than controls in all parameters assessed. Specifically, both ER and SR had larger LV wall and chamber

TABLE 1. Baseline demographics, clinical parameters, and training volumes in elite rowers (ER), subelite rowers (SR), and sedentary controls.

Demographics and Clinical Parameters	ER (n = 20)	SR (n = 20)	Control (n = 20)	P ^a Value
Age (yr)	25 ± 3*,†	20 ± 2	21 ± 2	<0.001
Height (cm)	197 ± 5*,†	186 ± 6†	177 ± 6	<0.001
Weight (kg)	98 ± 5*,†	82 ± 10	78 ± 9	<0.001
BSA	2.3 ± 0.1*,†	2.1 ± 0.2	2.0 ± 0.1	<0.001
Resting pulse (beats·min ⁻¹)	56 ± 7†	60 ± 5†	72 ± 8	<0.001
Systolic blood pressure (mm Hg)	124 ± 10	118 ± 11	120 ± 8	NS
Diastolic blood pressure (mm Hg)	62 ± 9†	58 ± 9†	72 ± 10	0.01
Family hypertension history	4/20	5/20	4/20	NS
Prescription medication use	8/20	5/20	7/20	NS
Cardiovascular medication	0/20	0/20	0/20	NS
Noncardiovascular medication	8/20	5/20	7/20	NS
Recent training volume (h·wk ⁻¹)	22 ± 6*,†	11 ± 4†	1 ± 1	<0.001
Endurance (h·wk ⁻¹)	19 ± 4*,†	9 ± 2†	0 ± 1	<0.001
Strength (h·wk ⁻¹)	3 ± 1	2 ± 1	0 ± 1	
Years prior competitive rowing	9 ± 4*,†	5 ± 2†	NA	<0.001

Values are presented as mean ± SD.

^a P value for one-way ANOVA across groups.

* P value < 0.05 for comparison with SR.

† P value < 0.05 for comparison with controls.

BSA, body surface area; HTN, hypertension; NS, not statistically significant, NA, not applicable.

dimensions, LV mass, left atrial volumes, and RV dimensions (Table 2). After BSA adjustment, all measurements remained higher in ER and SR than that in controls (Table 3).

Cardiac function. Functional parameters for the LV and RV are shown in Table 4. There were no significant differences in LV ejection fraction or LV mid-lateral wall longitudinal strain between either ER or SR and controls. In contrast, peak systolic tissue velocities in both the basal lateral LV wall and the interventricular septum were higher in both rower groups when compared with controls, with these differences reaching significance only for ER. Similarly, LV stroke volume, both unadjusted and BSA indexed, was higher in both rower groups than that in controls. All indices of RV systolic function were higher in both rower groups than that in controls.

Rowers had significantly enhanced LV and RV diastolic function when compared with controls. Specifically, parameters defining early LV diastolic function, including both peak transmitral E-wave and E' velocities, were higher in rowers than that in controls. Late diastolic indices, including peak A-wave and A' velocity, were also significantly higher in both rower groups compared with controls. The E/E' ratio was similar in all groups. In addition, both

rower groups had significantly higher E' and A' peak RV diastolic tissue velocities than controls.

Elite Rowers versus Subelite Rowers

Cardiac structure. ER had larger unadjusted LV wall and chamber dimensions, LV mass, left atrial volumes, and RV dimensions than SR (Table 2). After adjustment for BSA, LV and RV chamber dimensions and LV mass remained significantly higher in ER than that in SR. In contrast, BSA adjustment eliminated the differences in left atrial size and LV wall thickness.

Cardiac function. LV ejection fraction and peak LV systolic strain were similar in ER and SR. In contrast, peak systolic tissue velocities in both the basal interventricular septum and the basal lateral LV wall were higher in ER. Stroke volume, regardless of adjustment for BSA, remained higher in ER than that in SR. RV systolic function, as measured by fractional area change, peak systolic tissue velocity, and peak systolic strain, was higher in ER than SR.

With respect to LV diastolic function, there was no difference in peak E-wave or E' velocity between the two groups of rowers. In contrast, both peak A-wave and A' velocities were significantly higher among ER than SR. The

TABLE 2. Unadjusted cardiac structural parameters in elite rowers (ER), subelite rowers (SR), and sedentary controls.

Parameter	Reference Value ^a	ER (n = 20)	SR (n = 20)	Control (n = 20)	P ^b Value
LA volume (mL)	<59	75 ± 17*,†	62 ± 13†	46 ± 7	<0.001
IVS (mm)	<11	12.6 ± 0.9*,†	10.6 ± 0.7†	9.5 ± 0.8	<0.001
PWT (mm)	<11	12.7 ± 1.5*,†	10.5 ± 0.6†	9.6 ± 0.7	<0.001
LVEDV (mL)	67–155	171 ± 20*,†	140 ± 20†	125 ± 15	<0.001
LVESV (mL)	22–58	74 ± 12*,†	60 ± 8†	53 ± 9	<0.001
LV mass (g)	88–224	332 ± 45*,†	267 ± 31†	196 ± 23	<0.001
RVIDd (mm)	27–33	47 ± 4*,†	42 ± 5†	30 ± 5	<0.001
RV diastolic area (cm ²)	11–28	36 ± 3*,†	28 ± 4†	22 ± 5	<0.001
RV systolic area (cm ²)	7.5–16	18 ± 3*,†	15 ± 3†	13 ± 3	<0.001

Values are presented as mean ± SD.

^a Normal reference values adopted from the American Society of Echocardiography (11).

^b P value for one-way ANOVA across groups.

* P value < 0.05 for comparison with SR.

† P value < 0.05 for comparison with controls.

LA, left atrial; IVS, interventricular septal thickness; PWT, posterior wall thickness; LV, left ventricular; LVEDV, left ventricular end-diastolic volume; LVESV, end-systolic volume; RVIDd, right ventricular internal dimension at end diastole; RV, right ventricular.

TABLE 3. Cardiac structural parameters in elite rowers (ER), subelite rowers (SR), and sedentary controls after adjustment for BSA.

Parameter	Reference Value ^a	ER (n = 20)	SR (n = 20)	Control (n = 20)	P ^b Value
LA volume (mL·m ⁻²)	<29	32 ± 7†	32 ± 7†	24 ± 4	<0.001
IVS (mm·m ⁻²)	—	5.5 ± 0.3†	5.3 ± 0.5†	4.9 ± 0.3	<0.001
PWT (mm·m ⁻²)	—	5.4 ± 0.6†	5.4 ± 0.6†	4.9 ± 0.6	<0.001
LVEDV (mL·m ⁻²)	—	76 ± 6*,†	71 ± 8†	63 ± 7	<0.001
LVESV (mL·m ⁻²)	—	34 ± 4*,†	31 ± 5†	27 ± 4	<0.001
LV mass (g·m ⁻²)	<115	150 ± 11*,†	134 ± 16†	102 ± 13	<0.001
RVIDd (mm)	—	47 ± 4*,†	42 ± 5†	30 ± 5	<0.001
RV diastolic area (cm ² ·m ⁻²)	—	15.6 ± 0.9*,†	13.9 ± 1.5†	12.4 ± 2.4	<0.001
RV systolic area (cm ² ·m ⁻²)	—	7.5 ± 1.2†	8.0 ± 1.3†	6.0 ± 1.4	<0.001

Values are presented as mean ± SD.

^a Normal reference values adopted from the American Society of Echocardiography (11).

^b P value for one-way ANOVA across groups.

* P value < 0.05 for comparison with SR.

† P value < 0.05 for comparison with controls.

LA, left atrial; IVS, interventricular septal thickness; PWT, posterior wall thickness; LV, left ventricular; LVEDV, left ventricular end-diastolic volume; ES, end-systolic volume; RVIDd, right ventricular internal dimension at end diastole; RV, right ventricular.

E/E' ratio was similar in ER and SR. Similarly, RV early diastolic peak tissue velocity (E') was not different between the two groups, whereas late diastolic peak tissue velocity (A') was higher in ER.

DISCUSSION

Physiologic cardiac remodeling in response to both static and dynamic exercise training is well recognized. Careful inspection of previously published data defining this phenomenon reveals the presence of significant variability among athletes. Although several variables including athlete gender, ethnicity, sport/training type, age, and genetic profile have been shown to contribute to this variability, no single factor or combination of factors appears to explain all of the observed interindividual variability. Although the

magnitude of most physiologic adaptation and maladaptation is related to the degree of the stimulus responsible for the change, little is known about the dose-response relationship between exercise training and cardiac remodeling. To begin to address this area of uncertainty, we compared two groups of rowers (ER and SR) with significant differences in both short-term training volume and cumulative years of competitive rowing experience to one another and to sedentary controls.

Our findings can be summarized as follows. First, almost all cardiac parameters in ER and SR differed from those observed in sedentary controls, suggesting that significant cardiac remodeling was present in rower groups at both competition levels. Second, ER possessed larger LV chambers, LV mass, and RV chambers than SR even when chamber dimensions were adjusted for BSA. It is noteworthy

TABLE 4. Cardiac functional parameters in elite rowers (ER), subelite rowers (SR), and sedentary controls.

Parameter	ER (n = 20)	SR (n = 20)	Control (n = 20)	P ^a Value
LV systolic function				
Ejection fraction (%)	55 ± 6	56 ± 7	58 ± 8	NS
Longitudinal strain (%)	19 ± 4	21 ± 3	20 ± 4	NS
S' basal septum (cm·s ⁻¹)	7.8 ± 1.4*,†	6.9 ± 1.2	6.0 ± 1.0	0.008
S' basal lateral LV (cm·s ⁻¹)	8.7 ± 1.6*,†	7.1 ± 1.0	6.4 ± 1.1	0.006
Stroke volume	101 ± 18*,†	80 ± 17	73 ± 17	<0.001
Stroke volume/BSA (mL·m ⁻²)	47 ± 8*,†	41 ± 7	36 ± 8	0.03
LV diastolic function				
Transmitral E-wave (cm·s ⁻¹)	80 ± 12†	80 ± 16†	66 ± 7	<0.001
Transmitral A-wave (cm·s ⁻¹)	50 ± 9*,†	43 ± 10†	28 ± 6	<0.001
E/A ratio	1.6 ± 0.2*,†	1.9 ± 0.4†	2.4 ± 0.4	<0.001
E' basal septum (cm·s ⁻¹)	11.2 ± 1.7†	11.0 ± 0.9†	9.2 ± 0.9	<0.001
A' basal septum (cm·s ⁻¹)	5.0 ± 1.2*,†	4.2 ± 0.9†	3.3 ± 0.9	<0.001
E' basal lateral LV (cm·s ⁻¹)	11.8 ± 2.2†	11.9 ± 1.9†	9.8 ± 0.7	<0.001
A' basal lateral LV (cm·s ⁻¹)	4.2 ± 1.3*,†	3.2 ± 0.9†	2.1 ± 0.6	<0.001
E/E' ratio ^b	7.2 ± 2.0	6.8 ± 1.4	6.8 ± 1.1	NS
RV systolic function				
Fractional area change (%)	50 ± 7*,†	46 ± 8†	40 ± 6	<0.001
S' basal free wall (cm·s ⁻¹)	12.3 ± 2.1*,†	10.4 ± 1.7†	8.7 ± 1.6	<0.001
Mid free wall strain (%)	36 ± 7*,†	31 ± 6†	25 ± 3	<0.001
RV diastolic function				
E' basal free wall (cm·s ⁻¹)	9.8 ± 2.1†	10.3 ± 1.5†	7.6 ± 1.4	<0.001
A' basal free wall (cm·s ⁻¹)	6.6 ± 1.4*,†	4.3 ± 1.3†	3.1 ± 0.9	<0.001

Values are presented as means ± SD.

^a P value for one-way ANOVA across groups.

^b E/E' ratio was calculated using E' measured from the lateral LV wall.

* P value < 0.05 for comparison with SR.

† P value < 0.05 for comparison with controls.

LV, left ventricular; RV, right ventricular; S', peak systolic tissue velocity; E', peak early diastolic tissue velocity; A', peak late diastolic tissue velocity; E/E', transmitral E-wave velocity/peak early diastolic tissue velocity (E').

that adjustment for BSA eliminated the difference in LV wall thickness between ER and SR. Thus, the greater LV mass observed in ER was attributable to their relative larger LV chamber volumes. Third, resting RV systolic function, as assessed by several complimentary indices, was more enhanced in ER than that in SR. This finding is of particular interest given the fact that the RV's importance as a determinant of peak exercise capacity remains uncertain. Finally, ER demonstrated a marked enhancement of late diastolic filling in both ventricles when compared with SR.

The finding that rowing competition level is associated with specific cardiac attributes has two plausible explanations. First, the relative biventricular chamber enlargement, RV systolic function enhancement, and maximally efficient late diastolic filling may be innate characteristics of ER that pre-date exercise training and thus provide a selective advantage to perform at the highest level of this sport. Although prior data from childhood high-performers does not suggest that innate cardiac phenotype dictates success at a young age (24,25, 33,34,38), longitudinal studies characterizing cardiac structure from childhood through to the adulthood time of peak performance are needed to address this possibility. The second and more likely explanation is that the characteristic findings in ER may be a direct function of the amount of exercise training, both in the short-term and over cumulative years, to which these individuals are exposed. Several recent longitudinal studies demonstrating similar adaptations in rowers support this hypothesis (2,8). Further, long-term study, with a specific focus on the underlying cellular mechanisms that dictate myocardial remodeling in response to cumulative exercise exposure, is needed.

This study is the first to document the relationship between competition level and cardiac parameters in a direct comparative fashion. This finding has important implications with relevance to the clinical care of athletes and to the future exercise physiology research. For the clinician faced with differentiating healthy physiologic cardiac remodeling from that secondary to underlying disease, we provide clear evidence that competition level should be factored into the list of clinical variables currently recommended for this purpose (19). Further, our data clearly demonstrate that the majority of both elite and subelite rowers and likely similar caliber athletes from other endurance-based sports possess cardiac structural and functional values that fall far outside the range of what is currently considered normal (15). This finding underscores the need for the collaboration of the cardiovascular and sports medicine communities to establish data-driven reference ranges of normal for trained athletes. Our findings also provide the framework for future studies aimed at determining which cardiovascular parameters serve

as important determinants of exercise capacity (17,18). Confirmation of our findings coupled with definitive assessments of exercise capacity will lead to an improved understanding of how the heart and the circulatory system contribute to performance potential.

There are several limitations of this study. As previously addressed, the cross-sectional nature of our study design does not afford the opportunity to fully establish the causality of our observations. Although prior work suggests that our findings represent adaptation to variable volumes of exercise training, we cannot confirm that all of our observations can be explained simply by group differences in exercise exposure. Second, because all measurements were made with the athletes at rest and we were logistically unable to perform exercise capacity testing, we are unable to draw definitive conclusion about how our observations relate to cardiac function during exercise or to measures of exercise capacity. Third, as this study used athletes in real-world training environments, we were unable to characterize exercise training intensity using any of the usual quantitative metrics (exercise heart rate, power output, or percentage of peak oxygen consumption) during the period before study. Although our quantification of training volume ($\text{h}\cdot\text{wk}^{-1}$) demonstrated a "dose-dependent" relationship between cardiac remodeling and exercise training, our data do not permit us to determine whether group differences in exercise training intensity may have contributed to our observations. Finally, as ER had trained more ($\text{h}\cdot\text{wk}^{-1}$) in the immediate period before assessment and had accumulated more long-term rowing experience (yr) than SR, we are unable to draw definitive conclusions about the relative contribution of short- versus long-term training to the observed differences. Future work is warranted to determine which aspects of exercise training and sport participation are responsible for the competition level-associated differences we observed.

In conclusion, we present novel data documenting significant differences in cardiac structure and function between elite, Olympic caliber, and subelite university-level rowers. ER were found to have larger biventricular dimensions and greater enhancement of both RV systolic function and biventricular diastolic function than SR. These findings demonstrate that competition level is strongly associated with underlying heart structure and function and that this factor should be considered in both the clinical care and the future study of athletes.

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